

## CHYLOUS ASCITES IN LYMPHATIC LEUKEMIA\*

### REPORT ON TREATMENT WITH X-RAY

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THERE have been more than three hundred cases of chylous ascites recorded in the literature, and of these about five per cent have been due to involvement of the thoracic duct by one of the lymphoblastomata.

Blankenhorn,<sup>1</sup> in 1923, reported a mediastinal tumor causing chylothorax, which was treated by x-ray and radium. A year later the patient was living and seemingly well. In 1927, Ruddell<sup>2</sup> reported the treatment of a case of chylous ascites and chylothorax by radiation, but without any influence on the course of the disease. At section the thoracic duct was found to be obliterated by a large lymphogranulomatous mass, "probably of tuberculous origin." Cohn,<sup>3</sup> in 1931, noted the failure of radiation to alter the formation of chylous ascites in a case of lymphosarcoma.

The following case illustrates the temporary response which may follow radiation, and includes autopsy findings.

### REPORT OF CASE

Summary: A man with lymphatic leukemia who developed chylous ascites which disappeared temporarily after x-ray therapy; retroperitoneal mass obstructing the thoracic duct found at autopsy.

June 27, 1934.—F. B., a 67-year-old salesman of Italian descent, entered the medical service because of painless lumps in his neck, axillae, and groins, which had first been noticed about six weeks before. The past history was irrelevant. Examination showed him to be obese, but not apparently ill. On both sides of the neck, in both axillae, and in both groins were large, rather soft glandular masses. The spleen and liver were not felt. He was not anemic; the white count was 31,000, with 94 per cent lymphocytes. The urine was normal and the blood Wassermann negative. Fluoroscopy showed no tumors in lungs or mediastinum. A diagnosis of lymphatic leukemia was made after a biopsy of a cervical lymph node.

X-ray therapy was started at once, with irradiation of all of the glandular masses. Forty r were given daily for one week, then every four days for two months, and then two at fortnightly intervals, the last treatment being on October 10. A total of 920 r was given to each of six 15 centimeters round areas. The glands rapidly diminished in size and by the completion of the treatment could barely be made out. No anemia had developed and the white count had been reduced to 12,000, with 80 per cent lymphocytes.

October 24, 1934.—Patient reentered the hospital because his abdomen had become greatly swollen during the past three weeks, and he had lost strength and become dyspneic. Examination showed him to be cyanotic and to have an abdomen greatly distended with fluid. The glandular enlargements in neck, axillae, and groins had not recurred. The blood showed 3.8 million red cells, 84 per cent hemoglobin (Sahli), and 16,000 white cells, with 62 per cent lymphocytes. The colloidal osmotic pressure of the blood serum was 257 millimeters of water (moderately low).

\* From the Department of Medicine, Stanford University School of Medicine.

<sup>1</sup> Blankenhorn, M. A.: Chylous and Pseudo-chylous Effusions, *Arch. Int. Med.*, 32:129, 1923.

<sup>2</sup> Ruddell, K. R.: Chylous Ascites and Chylothorax Due to Lymphogranulomatosis of the Mesentery and the Receptaculum Chyli, *Indianapolis Med. J.*, 30:213, 1927.

<sup>3</sup> Cohn, I.: Chylous Ascites, *Am. J. Surg.*, 11:260, 1931.

Seven liters of chylous fluid were removed from the abdomen by paracentesis. This fluid was rose-pink in color, with an opaque, lustrous surface which suggested pink cream. It contained 1 per cent of fat and 4.4 per cent of protein. The sediment showed many red cells and lymphocytes. The liver and spleen were not palpable.

Since the chylous ascites was apparently due to leukemic infiltration along the thoracic duct, presumably with rupture of the duct, intensive x-ray therapy to the chest and abdomen was begun. Some 100 r were given daily for two weeks, the back and front being treated on alternate days. On November 5, while still being irradiated, two liters more of fluid were withdrawn from the abdomen. This was still opaque, but contained only 0.2 per cent fat and 4.5 per cent protein. When dismissed on November 12, he felt much stronger, had no ascites or glandular enlargements, and his blood showed 3.2 million red cells, 71 per cent hemoglobin, 4,000 white cells with 75 per cent lymphocytes.

January 6, 1935.—There was no evidence of re-accumulation of ascites.

January 24, 1935.—The patient reentered the hospital because his abdomen had again been swollen for a week. Nine liters of chylous fluid similar to that obtained at the first tapping were removed. The blood showed 3.2 million red cells, 73 per cent hemoglobin, and 3,000 white cells with 50 per cent lymphocytes.

During February, fifteen more liters of similar fluid were removed in two tapings, in spite of a series of three daily 100 r treatments to the trunk, and a second of eight identical treatments about three weeks later.

March, 1935.—He rapidly lost strength, and in a final paracentesis on March 16 seven more liters of the usual pink, milky fluid were removed. In this fluid there was a large amount of Bence Jones protein, as determined by the following method: The ascitic fluid was centrifuged to remove the free red cells (it contained no hemoglobin) and then diluted 1:10 with normal saline. Three drops of 20 per cent sulphosalicylic acid per cubic centimeter were added, with mixture boiled, and filtered while boiling. On cooling the filtrate, a precipitate appeared which redissolved on a second boiling. There was no Bence Jones protein in the urine. The blood on March 19 showed 3.2 million red cells, 70 per cent hemoglobin, 3,000 white cells with 51 per cent lymphocytes; the blood calcium was 7.8 milligrams per 100 cubic centimeters, the blood phosphorus 3.4 milligram per 100 cubic centimeters, and the phosphatase 2.3 units.

March 25, 1935.—The patient died. The *autopsy report*, abstracted by Dr. Alvin J. Cox, Jr., is as follows: The body showed moderate brown pigmentation of the skin. The peritoneal cavity contained about 900 cubic centimeters of turbid orange fluid, and both visceral and parietal peritoneal surfaces showed poorly defined areas of thickening, with scattered punctate hemorrhages. There was only slight enlargement of the peripheral lymph nodes. The largest, which was in the left inguinal region, measured three centimeters in diameter. In the retroperitoneal region, however, there was extensive lymph-node enlargement. Above the pancreas was a large mass of fused lymph nodes measuring 12 by 6 by 4 centimeters, and surrounding the abdominal aorta was a nodular collar of soft, markedly congested abnormal tissue about three centimeters thick. The vena cava passed through the lower part of this mass, but the receptaculum chyli and the lower portion of the thoracic duct could not be identified, though its normal position lay within the mass of tumor. The spleen was not enlarged, and no leukemic infiltrations of other organs were visible grossly.

Microscopical sections of the lymph nodes showed extensive infiltration of small lymphoid cells which in many places were sharply limited by the capsule of the nodes. The liver showed small areas of infiltration in the portal connective tissue. The bone marrow showed small focal collections of similar cells. Infiltration was not seen in the spleen.

Primary anatomical diagnosis: Leukemia, lymphatic.

## COMMENT

A case of lymphatic leukemia was complicated by chylous ascites, which was removed by paracentesis and did not reaccumulate for more than two months after x-ray therapy. Subsequently it recurred repeatedly, and autopsy showed obstruction of the thoracic duct by leukemic infiltration.

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## FAULTY POSTURE CONTRIBUTING TO GASTRIC AND DUODENAL ULCER

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THE writer has observed, over a period of years in medical practice, that gastric and duodenal ulcers occur more often in persons who sit a great deal, either at a desk or in faulty posture reading. The habit of sitting on the sacrum, rather than erect, is very common. This faulty posture, semi-reclining, pushes the costal arch inward, compressing the lesser curvature of the stomach and the cap of the duodenum, producing ischemia. Ischemia of the mucous membranes of the organs, with plenty of hydrochloric acid, would favor ulcer by allowing the bloodless tissue to digest its surface with resultant ulceration. It seems to me to be highly desirable that prolonged pressure should be avoided under the liver in this area during digestion. The frequency of ulcer in the lesser curvature of the stomach and the upper one-third of the duodenum would bear out this hypothesis.

It has also been my observation (both as a victim and with patients) that sitting erect and taking frequent deep breaths, if continued sufficiently long, has relieved the burning, gas, and distress.

I offer this as a most plausible contribution to the etiology, direct or indirect, to gastric and duodenal ulcer.

Bank of America Building.

## ACUTE APPENDICITIS ASSOCIATED WITH ACUTE MECKEL'S DIVERTICULITIS

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THE occurrence of acute inflammatory processes of Meckel's diverticulum, especially in children, is too well known to require recapitulation. In a symposium on "The Acute Abdomen," Miller and Wallace<sup>1</sup> have reviewed the literature on the anatomy and pathology of Meckel's diverticulitis, and have stressed the importance of searching for this condition when the appendix is not found to be acutely inflamed.

<sup>1</sup> Miller and Wallace: Meckel's Diverticulum in Acute Abdominal Emergencies, *Annals of Surgery*, 98:713 (Oct.), 1933.

In our search of the literature we have not found a report of the association of Meckel's diverticulum and the vermiform appendix, each in an acutely inflamed condition sufficient to give rise to the acute abdominal symptoms and at the same time each having a small area of surrounding intestinal and peritoneal inflammation in no way associated with the other.

## REPORT OF CASE

R. C., male, aged 32, had acute pain in the abdomen three days before admission to the Peninsula Community Hospital, which he attributed to indigestion due to hasty eating and fatigue from overwork. He was conscious for years of a small, annoying pain in his lower right abdomen, and felt that he "might have an appendix." On the evening before admission, he experienced very acute generalized abdominal pain, nausea and vomiting. In the morning, while at work, he called one of us. Examination revealed: Temperature, 100.4 degrees; pulse, 90; respiration, 16. The upper respiratory tract was negative. Lungs were clear and resonant, and the heart normal. There was moderate rigidity of the lower abdomen, especially of the lower belly of the right rectus. Pain only on deep pressure over McBurney's point. Rectal examination was negative. The leukocyte count was 26,500, with 82 per cent polymorphonuclear neutrophilic cells. The patient was immediately admitted to the hospital for surgery, with the diagnosis of acute appendicitis.

After spinal anesthesia, a McBurney incision was made and the rectus retracted. As the peritoneum was opened a slight amount of serous fluid exuded. After placing the retractors, a thumb-like projection was seen through the opening. This was a diverticulum about seven centimeters long, two centimeters in diameter at its base and attached to the small bowel on its antimesenteric border. It was exceedingly inflamed in its proximal half and dark, hemorrhagic-black at its tip. No opening could be found. After ligating, excising and burying the stump with a purse string suture, we found it was located about one meter from the ileocecal junction. The surrounding ileum was moderately injected in an area of about three inches. The appendix was then located and found to be markedly swollen, about ten centimeters long and curved upon its tightened mesentery. There was great engorgement of its blood vessels and reddish-black in its distal portion. It was removed in the usual manner and closure was made without drainage. The patient, discharged from the hospital on the eighth day, made an uneventful recovery.

## COMMENT

We are aware that, had the diverticulum not exposed itself immediately upon spreading the incision, we would have been content, having removed the acute appendix, and would probably have been acutely embarrassed at a later time with recurrence of symptoms remarkably simulating the appendicitis. Likewise, the hemorrhagic portion of the diverticulum might have been a peptic ulcer,<sup>2</sup> although no perforation could be found, and might have produced disastrous postoperative results had it been overlooked. This occurrence should stress the importance of further abdominal exploration, regardless of one's having found sufficient pathology to account for the surgical condition, since the percentage of diverticuli is from one to three per cent.<sup>3</sup>

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<sup>2</sup> Aschner and Karelitz: Peptic Ulcer of Meckel's Diverticulum, *Annals of Surgery*, 91:573 (April), 1930. Cobb: *Annals of Surgery*, 94:251 (Aug.), 1931.

<sup>3</sup> The Practitioner's Library of Medicine and Surgery, 4:577. D. Appleton, 1935.